bination with other factors present is a subject for conjecture.

The objections of Hodgson, Pugh and Young[3] to considering these bone lesions in diabetic patients and in other patients in their series as antedating the soft tissue injury which always accompanies them, apparently are well based. However, their denial of a disturbance of nerve supply, either somatic or autonomic, seems not well founded, since there appears to be abundant evidence, clinical and experimental, that damage to the nervous system can and does produce such lesions—whether primarily in the bone or in the soft tissue being beside the point. The Guillain-Barre type of albuminocytologic dissociation present in this and other cases reported may point toward some sort of radiculoneuronitis. At least it must be admitted that there is sufficient irritation of the central nervous system to produce pronounced elevation in protein content in the spinal fluid. Why, in the case herein reported, there should also have been a reversal of the albumin-globulin ratio is a mystery, unless one might postulate damage to the protein-forming function of the liver and reticuloendothelial system accompanying the diabetes of long standing.

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Ruptures of the Umbilical Cord with a Case of Intrapartum Rupture of All Three Vessels

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FOR practical purposes it would seem fitting to classify ruptures of the umbilical cord as (a) partial, and (b) complete. Under partial ruptures should be included:

1. Rupture of a varix of the umbilical vein.
2. Tearing of one or two of the three blood vessels.
3. Discontinuity of the epithelium covering the cord, with resultant extrusion of Wharton's jelly.

Bleeding from vessels in velamentous cord insertion is not uncommon. Partial ruptures have been discussed by Forsell,[5] and cases reported by a number of authors. In a case reported by Page,[7] a bleeding placental artery was tied prior to delivery and the child was saved.

Complete ruptures of the cord have been reported by Dyrenfurth,[4] Douglas,[3] Siddall[10] (first case), Baldi,[1] McClintock,[6] Bloxsome,[2] and Sackett.[9] Although the authors could find only these reports of a total of seven cases of complete cord rupture in the literature available in Honolulu, no doubt it is not too rare an occurrence.

ETIOLOGY

Among the causes of cord rupture are: (a) velamentous cord insertions, (b) absolute or relatively short (by wrapping about neck or extremities of fetus) cords, (c) localized necrosis in the cord, (d) hemangioma of cord vessels, (e) trauma due to podalic version, forceps blades, etc., in manipulative deliveries.

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ANATOMICAL CONSIDERATIONS

According to Spivack[11] there is some evidence that there are nerve fibers in the intra-abdominal portions of umbilical arteries. In addition, Spivack brings out the fact that the blood vessels of the umbilical cord differ anatomically from other vessels of similar caliber in the following ways: (1) The presence of folds and nodules of Hoboken in arteries and of semilunar folds in the vein, (2) the absence of true valves, (3) peculiar distribution of elastic tissue, (4) strongly developed arterial media whose powerful contractions are sometimes ascribed to the spiral or snail-like course of its muscle fibers, (5) absence of vasa vasorum and adventitia, (6) presence of a delicate connective-tissue stroma which contributes to the sponginess of the veins, (7) a nerve apparatus not yet positively demonstrated.

As a result of these various factors it is brought out that the umbilical arteries are almost impervious to the blood stream soon after birth. This seems to explain why Rachmanow,[8] in 1914, reported several thousand cases in which the umbilical cords were not tied and not a single newborn died from hemorrhage. These anatomical considerations will explain at least in part why at death there was seldom more than a small amount of intra-uterine bleeding despite severing of the umbilical arteries intrapartum. McClintock,[6] describing a case in which there was no bleeding from either end of the severed cord, concluded that the infant had died of asphyxia rather than hemorrhage. In several other cases also the amount of blood was minimal.

CASE REPORT

A 25-year-old primigravida was admitted to the Kapiolani Maternity Hospital Nov. 7, 1947. The estimated due date was Oct. 26, 1947. The course of the pregnancy had been uneventful prior to the onset of labor, and all laboratory tests revealed normal findings. Onset of labor was spontaneous, and the first stage was 15 hours and 20 minutes long. The second stage lasted 26 minutes. The fetal head engaged in the left occiput transverse position and apparently maintained this position until the cervix was fully dilated. Fetal heart tones had remained strong and regular until the fetal head reached to 2 cm. below the ischial spines. At this stage, the fetal heart rate rose suddenly and without warning to a rate too rapid to count and then suddenly ceased. No external bleeding was noted. The patient was draped quickly and under light cyclopropane anesthesia the fetal head was rotated easily from left occiput transverse to right occiput anterior position, using the Barton forceps. The delivery was completed by delivering the fetal head with Simpson forceps. Left mediolateral episiotomy was done. The fetal head was at plus 2 cm. below the ischial spines at the time of delivery and the cervix was fully dilated. Membranes were artificially ruptured just prior to the delivery. A large amount of meconium escaped along with amniotic fluid but a very small amount of dark red blood was present. The umbilical cord was looped once about the infant's neck and once in a complete turn around its right arm in the antecubital space. Where the cord crossed the antecubital space all three vessels—the two umbilical arteries and one umbilical vein—were completely severed. The cord was held together by a shred of epithelium not more than 0.5 cm. wide and infinitesimally thin. This was regarded as a complete severance of the cord inasmuch as the three vessels were totally cut through and the ends widely separated. There was no active bleeding in progress. The total amount of visible blood was not more than 5 cc. Trauma such as pinching of the cord by the forceps during delivery was ruled out by careful examination of the fetal head at the time of forceps application and prior to traction. A search was made for prolapsed cord and none was found.

Careful inspection revealed that the tear had occurred at a natural kink in the cord. This kink showed a lesser and a greater curvature and it would seem that the continued tension on the cord in late second-stage labor caused the shorter and inner portion of the kink in the cord to give way, shearing off the three umbilical vessels and leaving a narrow shred of the greater curve of the kink still holding.

It is believed that intra-uterine death occurred by asphyxia as it did in McClintock's[6] case, rather than by hemorrhage.

SUMMARY AND CONCLUSIONS

Partial severing of umbilical cord vessels has been reported fairly commonly in the medical literature.

We have been able to find reports of only seven cases of the severance of all three umbilical vessels during labor.

An eighth case of intrapartum avulsion of all three vessels is presented.

Etiology is discussed and attention is called to the role of actual and relative shortening of the cord in causing cord severance, especially if the cord contains kinks.

There is strong evidence that fetal death is more apt to occur from asphyxia than from hemorrhage, as blood loss is usually minimal when the vessels are completely severed.

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